AAMRL-TR-87-049

THE RELATIONSHIP BETWEEN CHANGES IN ARTERIAL PRESSURE, ESOPHAGEAL PRESSURE AND THE EMG OF VARIOUS MUSCLE GROUPS DURING THE L-1 STRAINING MANEUVER AT DIFFERENT SPINE-TO-THIGH ANGLES: A FINAL REPORT

CAROLL A. WILLIAMS, Ph.D.
J. E. DOUGLAS, M.D.
GARY MILLER, M.D.

QUILLEN-DISHNER COLLEGE OF MEDICINE, EAST TENNESSEE STATE UNIVERSITY

I. KARL

SYSTEMS RESEARCH LABORATORIES, INC.

R. L. WILEY

DEPARTMENT OF ZOOLOGY, MIAMI UNIVERSITY

A. R. LIND

DEPARTMENT OF PHYSIOLOGY, ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE

JUNE 1987

FINAL REPORT FOR PERIOD DECEMBER 1985-JUNE 1986

HARRY G. ARMSTRONG AEROSPACE MEDICAL RESEARCH LABORATORY
HUMAN SYSTEMS DIVISION
AIR FORCE SYSTEMS COMMAND

NOTICES

When US Government drawings, specifications, or other data are used for any purpose other than a definitely related Government procurement operation, the Government thereby incurs no responsibility nor any obligation whatsoever, and the fact that the Government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data, is not to be regarded by implication or otherwise, as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use, or sell any patented invention that may in any way be related thereto.

Please do not request copies of this report from the Armstrong Aerospace Medical Research Laboratory. Additional copies may be purchased from:

National Technical Information Service
5285 Port Royal Road
Springfield, Virginia 22161

Federal Government agencies and their contractors registered with Defense Technical Information Center should direct requests for copies of this report to:

Defense Technical Information Center
Cameron Station
Alexandria, Virginia 22314

TECHNICAL REVIEW AND APPROVAL

AAMRL-TR-87-049

The voluntary informed consent of the subjects used in this research was obtained as required by Air Force Regulation 169-3.

This report has been reviewed by the Office of Public Affairs (PA) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

FOR THE COMMANDER

Henning E. von Gierke

HENNING E. VON GIERKE, Dr Ing
Director
Biodynamics and Bioengineering Division
Armstrong Aerospace Medical Research Laboratory

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1a. REPORT SECURITY CLASSIFICATION: UNCLASSIFIED	1b. RESTRICTIVE MARKINGS
2a. SECURITY CLASSIFICATION AUTHORITY	3. DISTRIBUTION/AVAILABILITY OF REPORT: Approved for public release; distribution is unlimited
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE	
4. PERFORMING ORGANIZATION REPORT NUMBER(S): AAMRL-TR-87-049	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a. NAME OF PERFORMING ORGANIZATION: East Tennessee State University; 6b. OFFICE SYMBOL (If applicable)	7a. NAME OF MONITORING ORGANIZATION: HARRY G. ARMSTRONG AEROSPACE MEDICAL RESEARCH LABORATORY
6c. ADDRESS (City, State, and ZIP Code): Quillen-DISHNER, College of Medicine, Dept of Physiology, P.O. Box 19780A, Johnson City, TN 37614-0002	7b. ADDRESS (City, State, and ZIP Code): WRIGHT-PATTERSON AFB, OH 45433-6573
8a. NAME OF FUNDING/SPONSORING ORGANIZATION; 8b. OFFICE SYMBOL (If applicable)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER: F33615-81-C-0500
8c. ADDRESS (City, State, and ZIP Code)	10. SOURCE OF FUNDING NUMBERS

PROGRAM ELEMENT NO.	PROJECT NO.	TASK NO.	WORK UNIT ACCESSION NO.
62202F	7231	25	01

11. TITLE (Include Security Classification): The Relationship Between Changes in Arterial Pressure, Esophageal Pressure and the EMG of Various Muscle Groups During the L-1 Straining Maneuver At Different Spine-To-Thigh Angles: A Final Report

12. PERSONAL AUTHOR(S): Carole A. Williams, J.E. Douglas, Gary Miller, A. Karl, R.L. Wiley, A.R. Lind

13a. TYPE OF REPORT	13b. TIME COVERED	14. DATE OF REPORT (Year, Month, Day)	15. PAGE COUNT
FINAL	FROM DEC 85 TO JUN 86	87 JULY	46

16. SUPPLEMENTARY NOTATION

17. COSATI CODES		
FIELD	GROUP	SUB-GROUP
20	14	
06	04	

18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number): Flight Simulation; Stress (Physiology); (KR) L-1; Intrathoracic Pressure; Esophageal Pressure

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

Changes in arterial blood pressure, intrathoracic pressure, heart rate, and the electromyographic activity of various muscle groups were determined while 9 male subjects performed L-1 straining maneuvers at four spine-to-thigh angles (70, 84, 94, and 105°). There was no significant difference between the changes in these physiological variables due to the different angles. At the onset of the L-1, arterial pressure immediately increased to an average of 195 mm Hg but fell progressively during the next 5 sec of the maneuver, to 160 mm Hg. Arterial pressure remained at this level during the middle part of the L-1 and then recovered to 179 mm Hg by the end of the maneuver. Esophageal pressure followed essentially the same pattern of response, but heart rate progressively increased during the L-1. Electromyographic activity of all the muscle groups tested increased immediately at the onset of the straining, and then remained constant for the duration of the L-1. No one muscle group was utilized more than another. Inflation of an anti-G suit to 4 PSI had no effect on the variables measured. Keywords: G; Tolerance

20. DISTRIBUTION/AVAILABILITY OF ABSTRACT	21. ABSTRACT SECURITY CLASSIFICATION
☒ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT ☐ DTIC USERS	

22a. NAME OF RESPONSIBLE INDIVIDUAL	22b TELEPHONE (Include Area Code)	22c. OFFICE SYMBOL
R. E. van Patten	513-255-5742	AAMRL/BBS

DD Form 1473, JUN 86 — Previous editions are obsolete. — SECURITY CLASSIFICATION OF THIS PAGE

PREFACE

The experiments described in this report were carried out to determine 1) the changes in arterial blood pressure during the L-1 straining maneuver and the relative contribution to these changes by elevations in intrathoracic pressure, and whole-body muscular tensing and 2) the spine-to-thigh angle that would allow the greatest change in arterial pressure during the L-1 maneuver. This approach was used to evaluate the possible benefit that different spine-to-thigh angles might afford pilots under high +Gz stress.

The research was performed at the Quillen-Dishner College of Medicine, East Tennessee State University in the Department of Physiology under Contract Project F33615-81-C-0500, Systems Research Laboratories, Inc. and the Harry G. Armstrong Aerospace Medical Research Laboratory.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

METHODS

1. Subjects:

Ten male subjects volunteered to participate in this study. All were recruited from the graduate and medical student population of the College of Medicine. One of the original subjects was dropped from the study due to the discovery of Raynaud's phenomenon. All subjects were informed of the purposes and procedures of the experiments according to the regulations of HHS and the Institutional Review Board of East Tennessee State University. Each subject signed an Informed Consent Form, and these were placed on file in the Department of Physiology. There were no untoward effects resulting from any of the procedures carried out in this study.

A summary of anthropometric information about the subjects is given in Table 1 (see page 29).

Subjects completed a brief training period, lasting 2 weeks, during which they learned to perform the L-1 straining maneuver, so that each effort would be consistent, and to perform sustained fatiguing isometric contractions of the hand-grip. Each subject exerted 2 maximum voluntary contractions (MVC) on a hand-grip dynamometer with a 3 minute recovery period between each maximum effort. The higher tension of the two trials was taken as the MVC. Subjects then exerted 3 consecutive fatiguing contractions at 40% MVC with 3 min resting periods between each contraction. Fatigue was designated at that time when tension could not be maintained on the target, even with a maximum effort.

2. Chair Apparatus:

All experiments were performed in a version of a pilot's seat that had a movable foot platform and movable back so that changes could be made in the angle between the spine and the thigh and the seatback (see Figure 1, page 30). Subjects were seated in the chair with their backs against the seatback and

restrained in this fixed position by a lap belt. A movable footplate was adjusted for each subject so that a neutral thigh-to-calf angle of 105-110^{o} was achieved. The length of foot-plate required for each of the subjects is listed in Table 1. Subjects indicated this angle felt optimal for both prolonged restrained sitting and for leverage in generating force by the legs. The latter proved unnecessary for this particular study since subjects were instructed not to generate any external net force by their legs during the L-1 maneuver. A force transducer was connected to the foot platform to detect the angular force generated against the bottom of the platform. This force was measured throughout the experimental period to detect any alterations during the straining in comparison to the resting periods. The entire foot platform was raised or lowered to achieve spine-to-thigh angles that averaged 70^{o}, 84^{o}, 94^{o} and 105^{o}. All but one of the experiments reported here were conducted with the seatback at a 30^{o} angle with respect to a perpendicular plane to the floor (see Figure 1). A further series of experiments was conducted at a spine-to-thigh angle of 105^{o} with the seatback lowered to 60^{o} to determine whether this would affect the EsoP and BP generated during the straining.

3. Physiological Measurements:

a) ECG Standard LL I configuration was used to record the electrocardiogram from subjects on a Grass Model 7 polygraph for 30 sec prior to, during the 15 sec L-1 maneuvers and for 15 sec following the maneuver on each subject at each of the different spine-to-thigh angles. Heart rates (HR) were calculated from 5 sec intervals during these segments and expressed as beats.min^{-1} (bpm).

b) Electromyography (EMG): The EMG was recorded from the surface of the skin over the following muscle groups: right intercostals between the 5th - 6th intercostal spaces; the right lower abdominal quadrant at McBurney's point (one-

third the distance from the anterior superior iliac spine to the umbilicus); center of the left quadriceps; center of the right biceps; center of the right calf muscles; and the right pectorals. The skin was prepared by scrubbing the surface with acetone. Self adhesive silver-silver chloride electrodes were placed over the designated muscle groups, electrode leads were attached and covered with non-allergenic surgical tape. This kept the electrodes secured throughout the experiment. Care was taken to place the electrodes in the same spot during repeated experiments. Electrodes were attached to a pre-amplifier (Teca Model PA62T) and then connected into an electromyograph (Teca Model M). The raw EMG signal was displayed on an oscilloscope and simultaneously fed into a root mean squared (rms) converter (Analog Devices). The integrated signal was recorded on a Fisher Recordall. The sites for measurement were assigned number designations and during the course of the experiment only the number designation was used for identification purposes. This helped to eliminate any bias on the part of the subject to concentrate on any one particular muscle group. Subjects were instructed to remain quiet and relaxed prior to the generation of L-1 maneuvers. Since the EMG signal was negligible during these resting periods, it was taken as the baseline and set at zero signal height. The change in muscular activity due to tensing was then measured as the change in the height (in cm) of the integrated EMG signal. Typically, a 1 cm deflection was equivalent to 7.8 uvolts.

c) Intraesophageal Pressure (Peso): Intraesophageal pressure was measured from inflated esophageal balloons attached to a pressure transducer and taken to be a reflection of the changes that occurred in intrathoracic pressure. Respiratory balloons were secured to a length of polyethylene tubing (PE205) with surgical suture. The end of the tubing covered by the balloon was perforated with a series of 1-2 mm diameter holes to ensure proper transmission of pressure

changes. The appropriate length of tubing was marked for mid-thoracic positioning and subjects swallowed the balloon-catheter assembly by first inserting it through the nasal opening. Once the balloon was swallowed and placed at the marked length, and after the subject was seated in the "pilot" chair, the assembly was attached to a Statham-Gould pressure transducer (P23b). The pressure signal was recorded on a Grass Model 7 Polygraph. Baseline was checked electronically with the balloon deflated and after inflating the balloon with 13 ml of air through a ground-glass syringe. The position of the tip of the catheter was verified by a negative intraesophageal pressure during quiet breathing, and by a more negative deflection (downward) during inspiration and less negative (upward) deflection during expiration. Peso was recorded for the entire duration of the experimental period. Subjects kept a nose-clip on during the measurement periods which helped to reduce movement of the catheter due to spontaneous esophageal waves. Peso was measured during the 30 sec prior to, during the 15 sec L-1 maneuvers and for 15 sec following the maneuvers. Values for Peso were averaged from 2-3 breaths during the rest and recovery periods and measured at the start and end of each segment of the 15 sec L-1 maneuver (i.e., at the 0 sec and 5 sec mark, at the 6 sec and 10 sec mark, and at the 11 sec and 15 sec mark of the L-1 maneuver).

d) Arterial Blood Pressure (BP): Direct measurement of arterial blood pressure was made through a catheter inserted percutaneously into the radial artery. Sterile procedures were followed by first cleansing the catheterization field with alcohol and then by scrubbing the area with a betadine solution. The area was anaesthetized by a subcutaneous injection of 2% lidocaine. A Medi-Cut teflon catheter unit was filled with sterile heparinized saline and inserted into the radial artery percutaneously using a 20g needle. Once entry was made into the artery, the needle was withdrawn and the catheter was connected to a

constant-flow sterile heparinized saline drip (3 ml/hr) and to a Statham-Gould pressure transducer (P23b) by way of a sterile 3-way stopcock. Baseline for the pressure signal was positioned prior to the insertion of the catheter with the fluid-filled system and calibration of the catheter-transducer assembly was made prior to the insertion of the catheter. Once the catheter was inserted, the calibration was rechecked to assure that the baseline had not shifted. The baseline was checked a final time at the end of the experiment for each subject. The catheter was secured in place with non-allergenic surgical tape and the hand and lower arm were fixed to a styrofoam support. The pressure pulse was recorded continuously during the experimental period on a Grass Model 7 polygraph and measurements of BP were taken 30 sec prior to, during the 15 sec L-1 maneuver and 15 sec following the maneuver. Mean arterial blood pressure (MBP) was calculated from the diastolic plus one-third of the pulse pressure.

4. Experimental Protocol:

a) Experiment 1 - Part A: On the day of the experiment, the sets of ECG and EMG electrodes were applied to the subjects. Subjects put on an anti-G suit and then inserted the respiratory balloon. The subject was positioned into the "pilot's" chair with the seatback fixed at an angle of 30° and the thigh to leg angle at 110°. The intra-arterial catheter was inserted into the left radial artery. Subjects rested for 15 min before beginning the series of L-1 maneuvers. During the last 30 sec of this period, ECG, EMG, Peso and BP were recorded for measurement of these parameters. Subjects were then instructed to exert the L-1 maneuver with whole body tensing for 15 sec with the foot-platform positioned so that the spine-to-thigh angle was either 70°, 84°, 94° or 105°. For the L-1 maneuver, subjects were given a count-down, instructed to initiate the straining and at the 5 sec and 10 sec mark to take a breath and to generate a straining effort again. They were given a signal to stop the maneuver at the

15 sec mark. Observation of the subjects assured that each effort after the breath at 5 and 10 sec was consistent with the first. Subjects were instructed and reminded to keep the left arm (with the arterial catheter) as relaxed as possible to avoid any artifact in the pressure pulse tracing that might result from muscular tensing of that arm. The order of the spine-to-thigh angle was randomized. In addition, the order of recording EMG from the various muscle groups was randomized and as explained above, only numbered designated sites were used for identification purposes. A variable resting period was allowed following the completion of a straining maneuver to permit BP to return to resting conditions before the next straining maneuver at a different angle was performed. A flow diagram illustrating the procedures used for these experiments is shown in Figure 2. The matrix illustrating the order for the various spine-to-thigh angles is given:

	Spine-to-Thigh Angle
Condition A	70°
Condition B	84°
Condition C	94°
Condition D	105°

	PART A				PART B			
	1	2	3	4	1	2	3	4
Subject (A)	B	D	A	C	A	C	B	D
(B)	A	C	B	D	C	A	D	B
(C)	D	A	C	B	B	D	C	A
(D)	C	B	D	A	D	B	A	C
(E)	A	D	B	C	B	C	D	A
(F)	C	B	D	A	D	A	C	B
(G)	D	C	A	B	A	D	B	C
(H)	D	A	C	B	C	B	A	D
(I)	D	A	C	B	C	B	A	D

Part B: Prior to starting the straining maneuvers, subjects were instructed to remain relaxed and the anti-G suit was inflated for 15 sec at 4 PSI. This was done to determine what effect inflation of the suit would have on HR and BP. The same procedure was followed for Part B of the experiment as

for Part A as described above, with the exception that upon the onset of the L-1 maneuver, the anti-G suit was simultaneously inflated to 4 PSI from a compressed gas tank. The suit remained constantly inflated at this pressure for the entire 15 sec period and was simultaneously deflated when the subject was instructed to relax (see Figure 2).

At the completion of the 4 spine-to-thigh angles, subjects were then asked to perform the following control procedures: 1) the arm containing the arterial catheter was tensed for 15 sec to the same extent (as assessed by the EMG) that subjects tensed the rest of their muscles during the L-1 maneuver. The subjects were instructed to talk to the observers so that no breath holding or chest fixing could occur. This experiment was intended to determine whether arm tensing would itself alter the BP or in any way alter the signal recorded. 2) Whole body tensing was performed for 15 sec with the same degree of effort that was generated during the L-1 maneuver with the exception that subjects were instructed to talk to the observers to minimize breath-holding or chest-fixing. The intention was to determine what effect muscle tensing without increases in intrathoracic pressure would have on BP. 3) Subjects were instructed to perform a Valsalva maneuver for 15 sec using the same starting inspiratory effort they did for the L-1 maneuver without any body tensing. The intention was to determine what effect positive intrathoracic pressure for 15 sec would have on HR and BP.

Following a 5-10 min recovery period, the foot-platform was positioned for the 105° angle and the seatback altered to 60° and the subject exerted an L-1 maneuver as described above.

b) Experiment 2: Part A For this experiment, all subjects were prepared as described above for the protocol in Experiment 1. However, subjects exerted 2 maximum voluntary contractions (MVC) using a hand-grip dynamometer before they

began their preparation and set-up for the straining experiment. They repeated the procedure followed as described for Experiment 1 Part A with a different randomized order for the spine-to-thigh angles. The matrix describing this was as follows:

	PART A				PART B			
	1	2	3	4	1	2	3	4
Subject (A)	D	B	A	C	B	D	C	A
(B)	B	C	D	A	C	B	A	D
(C)	C	A	B	D	A	C	D	B
(D)	A	D	C	B	D	A	B	C
(E)	D	A	C	B	C	B	D	A
(F)	A	D	B	C	D	C	A	B
(G)	B	D	A	C	A	C	B	D
(H)	C	B	D	A	B	A	C	D
(I)	C	A	D	B	A	D	C	B

At the completion of the fourth angle, subjects were then instructed to perform a sustained hand-grip contraction at 40% MVC to fatigue. Four of the subjects (A, C, D and G) participated in preliminary experiments in which they simultaneously exerted hand-grip contractions at either 30, 40 or 50% MVC during the L-1 straining maneuvers to determine whether BP would respond differently when isometric muscle contractions at low to high tensions were applied.

Part B: The same protocol was followed for inflation of the G-suit as described for Experiment 1, and the same 4 subjects additionally exerted simultaneous hand-grip contractions. At the completion of the fourth angle, subjects again exerted a sustained hand-grip contraction at 40% MVC to fatigue.

5. Data Analysis:

The values measured for Peso, HR, MBP and EMG were averaged for each subject for Part A of both experiments and for Part B of both experiments, with the exception of the 4 subjects who performed the preliminary procedure involving simultaneous hand-grip and L-1 maneuvers. An average of each of the

physiological parameters was made from the 9 subjects for Part A and for the 6 subjects who did not perform simultaneous handgrip contractions during Part B. Values given are the means + SEM. Significance was determined from analysis of variance for paired data for the changes in these parameters during the course of the L-1 straining maneuver and between the different angles. Student's T test for paired data was used to determine the level of significance between Part A and Part B. Significance was achieved when $P<0.05$. The relationship between changes in Peso and MBP was determined from regression analysis for the individual pressure measurements for each subject for Part A and B. Significance was achieved when a correlation coefficient was 0.5 or higher. Computer generated diagrams for this analysis are given in Figure 7.

RESULTS

A section of the recording measuring force applied to the foot platform, ECG, BP and Peso is shown in Figure 3. This demonstrates the typical changes in the above parameters occurring in response to an L-1 straining maneuver applied at one of the spine-to-thigh angles with the seatback at 30°. The onset of the maneuver, indicated by the marker shown in the time panel, was associated with the abrupt positive increase in Peso and BP. There was no significant force applied to the foot platform during the L-1 maneuver. Heart rate increased progressively over resting levels from the beginning to the end of the straining maneuver and then slowed during the first 15-20 sec of the recovery period immediately after the release of the L-1 maneuver. This was characteristic for the majority of the subjects. The arterial blood pressure increased dramatically at the onset of the straining maneuver, but during the next 4-5 sec, steadily declined until the subject took a breath and applied the straining maneuver again. The BP remained fairly constant during the 6^{th} - 10^{th} sec of the maneuver, as indicated, and then showed the same pattern during the 11^{th} - 15^{th} sec as it did during the first 5 sec of the maneuver. Pulse pressure decreased during 0 - 5 sec from resting values, remained essentially the same for the remainder of the maneuver and then significantly increased during the first 15 sec of the recovery period after the release of the maneuver, as can be seen in the tracing. This was due to an increase in the systolic blood pressure and a decrease in the diastolic blood pressure. Esophageal pressure remained at or slightly below atmospheric (taken as 0 mm Hg) during quiet breathing at rest. It decreased (i.e., closer to 0 mm Hg) during expiration and increased (i.e., became more negative) during inspiration. Esophageal pressure became abruptly positive at the onset of the L-1 maneuver as the subjects attempted to expel air against a closed glottis with whole body tensing. Even though the straining effort was maintained, Peso fell during the three segments of the L-1 maneuver,

but remained positive. Esophageal pressure returned to resting levels within one to two breaths at the release of the straining effort.

1. Esophageal Pressure:

The peak intraesophageal pressure (intrathoracic) achieved without an inflated G-suit at the onset of the L-1 maneuver averaged 117 ± 5 mm Hg as shown in Figure 4A. Within the first 5 sec of the straining, the pressure fell by 34 mm Hg to average of 83 ± 3 mm Hg ($P<0.01$). Following the breath taken at the 5 sec mark, pressure recovered to about 100 mm Hg but then progressively fell during the next 5 sec by 25 mm Hg. The same pattern was seen for the last 5 sec of the maneuver (i.e., from 11 - 15 sec) with the resulting Peso averaging 70 ± 1 mm Hg. The changes in Peso were unaffected by the different spine-to-thigh angles.

The peak Peso achieved at the onset of the L-1 maneuver with the G-suit inflated averaged 103 ± 6 mm Hg and this declined within the first 5 sec of the maneuver by 20 mmHg to 83 ± 10 mm Hg (P 0.05), as shown in Figure 4B. The same pattern of change in Peso occurred during the straining maneuvers with the G-suit inflated as it did without the G-suit inflated. There was no significant difference in the peak Peso achieved at the onset of the L-1 maneuver with or without the G-suit inflated (P 0.05), nor between any of the Peso measured when the G-suit was inflated.

The Peso returned to resting levels quickly after the maneuver was ended. The decline in Peso during the 5 sec segments of the L-1 maneuver was not due to lack of effort by the subjects, as indicated by the maintenance of the EMG during the maneuver (see Figures 8-10). The decline in the Peso during the 5-sec segments of the L-1 maneuver is similar to the changes in Peso which were measured during a Valsalva maneuver with no body tensing (see Figure 14).

2. Heart Rate

Heart rates increased steadily during the straining maneuver by about 50 bpm from resting levels of 80 bpm to peak levels of 132 bpm at the end of the straining effort, as shown in Figure 5. There was no effect on the HR attained with the inflation of the anti-G suit, as shown in Figure 5B and the changes in HR were independent of the spine-to-thigh angle. The HR at the end of the straining maneuver was significantly higher than at rest ($P<0.001$). Heart rate declined to 102 $\pm$ 5 bpm with the G-suit deflated and to 112 $\pm$ 7 bpm with the G-suit inflated ($P>0.05$) 15 sec after the L-1 maneuver.

3. Arterial Blood Pressure

The changes in mean arterial blood pressure (MBP) are shown in Figure 6. The resting MBP was 115 $\pm$ 2 mm Hg (Part A) and 108 $\pm$ 3 mmHg (Part B) before the start of the straining maneuvers. This was higher than the MBP measured by auscultation during resting periods on training days, which averaged 94 $\pm$ 2 mm Hg. This difference ($P<0.05$) was probably due to the apprehension associated with the experimental protocol for these experiments; this was the first time most of the subjects swallowed an esophageal balloon and none of the subjects previously had undergone arterial catheterization.

As can be seen in Figure 6, the changes in MBP were independent of the spine-to-thign angle. The peak MBP occurred at the onset of the straining maneuver, averaging 195 $\pm$ 5 mm Hg with the G-suit deflated and 187 $\pm$ 3 mm Hg inflated. These values were not significantly different ($P> 0.05$). Pressure fell by 30-35 mm Hg to an average of 162 mm Hg (G-suit deflated) and to 158 mm Hg (G-suit inflated) respectively, during the first 5 sec of the L-1 maneuver. This decrement was significant ($P< 0.05$) for both conditions. The MBP remained at this level, averaging 160-165 mm Hg for the next 5 sec of the maneuver and then recovered to levels near 180 mm Hg for the last 5 sec of the maneuver.

The fall in the MBP during the initial part of the maneuver (i.e., 0-5 sec) resembles the changes in MBP during a Valsalva maneuver (see Figure 14). This pattern is indicative of impeding the venous return due to the high positive intrathoracic pressure, a fall in cardiac output and thus the decrease in arterial pressure and the compensatory reflex increase in HR (see Figure 5). The pattern during 5-10 sec and 10-15 sec of the maneuver may be due to two mechanisms. First, the breath allows for some venous return from the periphery and second, there may be some compensatory shift of blood volume from the pulmonary circulation into the left heart. This compensatory shift would not occur immediately upon the initiation of the L-1 maneuver, but later, probably by the 10-15 sec period. The suggestion that venous return is compromised during the L-1 maneuver is supported by the changes in the pulse pressure (the difference between systolic and diastolic blood pressures) which occurred. While pulse pressure averaged 57 $\pm$ 8 mm Hg at rest, it decreased to 49 $\pm$ 7 mm Hg by the end of the maneuver, but then increased significantly over resting values ($P<0.05$) to 83 $\pm$ 12 mm Hg 15 sec after the release of the straining maneuver. The recovery period (at 15 sec) was also characterized by a marked decrease in heart rate (see Figures 2 and 5). These two cardiovascular parameters indicate increases in venous return upon release of the straining maneuver, allowing for enhanced filling of the heart. This response is manifested by the increasing systolic pressure, signifying the greater stroke volume, and a fall in diastolic pressure, indicating withdrawal of sympathetic tone.

While the inflation of the anti-G suit to 4 PSI at rest caused no change in HR or Peso, there was a 20 mm Hg increase in MBP at the onset of the inflation (see Figure 11). This effect was evidently insufficient to counteract the effects of high intrathoracic pressure on the impedance of venous return. The data in Figure 7 indicate the relationship between changes in Peso (intrathoracic pressure) and arterial blood pressure. The relationship, as

described from the equations listed in Figure 7, was not affected by inflation of the G-suit. The correlation coefficient for the relationship between MBP and Peso when the G-suit was deflated was 0.518. Student's T was found to be significant ($P<0.001$). The regression analysis for the relationship between MBP and Peso when the G-suit was inflated yielded a correlation coefficient of 0.460 and Student's T test also indicated significance ($P<0.001$). Both analyses indicated (from the slope of the lines) there was a relatively small increase in the MBP for a given change in Peso. When data were analyzed for the relationship between MBP and Peso at individual spine-to-thigh angles, similar regression equations resulted. At 70°, $y = 0.27 X + 151.1$, $r = 0.454$; at 84°, $y = 0.32 X + 145.9$, $r = 0.499$; at 94°, $y = 0.38 X + 139.7$, $r = 0.525$; and at 105°, $y = 0.25 X + 148.5$, $r = 0.368$. These data indicate that Peso will contribute to the change in MBP during the L-1 maneuver.

4. Electromyographic Activity

The EMG, integrated for the root mean square of the signal, indicated that all six muscle groups tested, pectorals, biceps (Figure 8), intercostals, abdominals (Figure 9), quadriceps and calf (Figure 10), contributed to the whole body tensing during the L-1 maneuver. As with the other physiological parameters measured, the changes in EMG activity were independent of the spine-to-thigh angle. All muscle groups showed the same response. Upon the onset of the L-1 maneuver, when muscle tensing was initiated, the EMG signal increased abruptly and then remained constant for the 15 sec maneuver. This indicated that no one muscle group was fatiguing. The change in the EMG signal was similar for all muscle groups, with the exception of the signal recorded from the quadriceps, and the biceps. These signals were slightly higher, though not significantly higher than the others and may be due to the larger muscle mass in the case of the quadriceps and their proximity to the surface of the body. The

similarity of the EMG recorded from these various muscle groups indicates that all contributed fairly equally to the tensing action during the L-1. The consistent signal throughout tne duration of the L-1 indicated that the effort, due to muscle tensing, remained constant for the entire straining maneuver. This is important since it indicates that the fall in Peso and MBP were not due to a decrement in the straining effort made.

5. Physiological Changes During Tensing without L-1

In order to determine whether the blood pressure signal recorded would be altered, and thus affect the results reported by any muscular tensing of the arm containing the arterial catheter, subjects were instructed to specifically tense this arm to the same extent they tensed the rest of their body during the L-1 maneuver. There was no damping of the pressure pulse as a result of this procedure. Figure 12 shows the changes in HR and MBP that occurred in response to arm tensing alone. Heart rate increased by 13 bpm ($P>0.05$) while MBP increased by 13 mm Hg upon the start of the arm tensing ($P<0.001$), and remained elevated above the resting value by some 10 mm Hg ($P<0.02$). This procedure indicated that the technique used to record arterial pressure was not affected by muscular tensing that may have occurred during the 15 sec straining maneuver, and that the decrement in blood pressure seen especially during the period 0-5 sec of the L-1 maneuver was not due to a damping of the signal.

Since changes in Peso in and of themselves will affect the arterial blood pressure, it was important to determine what contribution to the elevation of blood pressure during the L-1 maneuver was due to low tension muscular contraction of the entire body. In order to determine this influence, subjects were instructed to tense their bodies as they did during the L-1 maneuver, but they were required to talk to the observers so that only limited chest fixation or increases in Peso could occur. The results of this procedure are shown in

Figure 13. Whole body tensing increased HR by some 35 bpm above resting levels ($P<0.001$) by the end of the 15 sec period. This is in comparison to the 50-60 bpm increment in HR during the L-1 maneuver. The change in HR mediated by increased sympathethic tone during tensing may be due to the increased metabolic rate that occurs in response to this low level muscular activity, and the alterations in local blood flow that must occur in response to this. The MBP increased by some 35-40 mm Hg ($P<0.001$) as a result of whole body tensing. This is in comparison to the 85-90 mm Hg increment in MBP seen initially in response to the L-1 maneuver.

6. Physiological Changes During Valsalva

It became clear that the L-1 maneuver was imposing a complex reflex response on the cardiovascular system that partially resembled the events that occur during a Valsalva maneuver. Subjects were instructed to exert a Valsalva maneuver for 15 sec without the whole body tensing that accompanied the L-1 maneuver to determine whether the changes in HR and BP would be similar. The results of this procedure are shown in Figure 14. The Peso generated at the onset of the Valsalva (84 $\pm$ 10 mm Hg) was similar to the intrathoracic pressure generated at the start of the L-1 maneuver. The Peso fell by some 40 mm Hg during the Valsalva which was similar to the 35 mm Hg pressure drop measured during the L-1 maneuver. Mean blood pressure increased by about 55 mm Hg at the onset of the Valsalva and then declined by 35-40 mm Hg by the end of the maneuver. This pattern was quite similar to that described for the pressure changes in response to the L-1 maneuver. Similarly, the HR increased progressively during the Valsalva, just as it did during the L-1 maneuver.

These data indicate that the cardiovascular system is responding to the L-1 maneuver in a manner that is quite similar to the adjustments made in response to a Valsalva stress. If the changes in MBP due to whole body tensing and the

inital changes seen in response to the Valsalva are combined, then the transitory changes in MBP that occur during the L-1 maneuver can be accounted for.

7. Physiological Changes During L-1 Maneuver at 60° Seatback

Subjects performed the L-1 maneuver at a spine-to-thigh angle of 105° with the seatback at 60° to vertical (see Figure 1) to determine whether configuration of the seatback would alter the physiological responses to the L-1 maneuver. As can be seen from Table 2, there was no significant difference in the Peso, HR or MBP achieved during the L-1 with the chair in this configuration when compared to the levels achieved with the subject in a chair with a 30° seatback.

8. Arterial Blood Pressure During the L-1 and Isometric Handgrip Exercise

The data resulting from the specified protocol indicated that the potential contribution to the elevation of MBP by high tension muscular contraction was not being utilized effectively when instructing pilots simply to tense their bodies. Preliminary experiments were conducted on 4 of the subjects during the repeat experiments during which they simultaneously applied the L-1 maneuver and handgrip isometric contractions at 30%, 40% and 50% MVC. As indicated in Table 3 and Figure 15, applying isometric handgrip contractions at low tensions, 30% and 40% MVC, during the L-1 were ineffective in maintaining or elevating MBP above the levels seen in response to the L-1 alone. Performing isometric handgrip contractions at 50% MVC however, were effective in maintaining and during the latter stages of the L-1 maneuver, in elevating the MBP significantly. Figure 15 shows that this amounts to an average of 25 mm Hg higher than the MBP during the L-1 alone. If similar blood pressures were achieved under G-stress, this would allow tolerance of 1 G more than can be achieved with the L-1 maneuver alone.

In one subject,who initially performed handgrip contractions at 70% MVC with 15 sec L-1 maneuvers, the MBP followed this pattern:

	Rest	0-5 sec	5-10 sec	10-15 sec	Recovery
mm Hg	118	197→152	148→181	208→232	127

In this same subject, the pattern of blood pressure during the L-1 maneuver alone followed:

mm Hg	109	180→137	151→138	180→170	111

This suggests that the higher the tension and thus the quicker the onset of the pressor response due to muscle contractions, the greater is the additive (or possibly a synergistic) effect of simultaneous isometric muscle contraction and the L-1 maneuver in the maintenance of arterial blood pressure.

DISCUSSION

The main findings of this study address questions regarding changes in pilot seat configuration and how these differences might affect +Gz tolerance which occur during aerial combat maneuvers. We found that altering pilot configuration from a more closed orientation, with a spine-to-thigh angle of 70° with knees closer to the chest, to a more open position, with a spine-to-thigh angle of 105°, did not affect the ability of the L-1 maneuver to elevate arterial blood pressure at 1 G, when subjects were seated at a 30° seatback angle. We also found that placing the subjects in a more supine position, with the seatback at a 60° angle, did not affect the blood pressures generated in response to the L-1 maneuver.

This study also attempted to determine the relative contribution to the elevation in blood pressure attributable to changes in intrathoracic pressure and to whole-body muscular tensing. Experiments were performed with and without the use of an anti-G suit.

It is well documented that maneuvering a high performance aircraft places extreme stress on the cardiovascular system and that physiological limitations of the cardiovascular system may be exceeded by the performance capabilities of the aircraft. Exposure to repeated and sustained acceleration forces in the head-to-foot direction (+Gz) can be made more tolerable by the use of protective devices such as the anti-G suit and the performance of protective straining maneuvers such as the L-1 and M-1. The L-1 maneuver, which was examined in this study, uses tensing of abdominal and whole body skeletal muscles with a Valsalva maneuver against a totally closed glottis (1).

The measurement of the changes in intrathoracic pressure (intra-esophageal pressure), arterial blood pressure and heart rate during L-1 maneuvers indicate that even within the 5-sec periods of sustained Valsalva action, compromises are

made by the cardiovascular system which may not afford the pilot adequate acceleration protection, if that acceleration profile is sustained longer than 5 sec and consists of increasing +Gz forces over that period of time. The peak mean blood pressure was acheived at the onset of the L-1 maneuver, as indicated in Figure 6, and amounted to 195 ± 5 mm Hg with the subjects in an upright position (seatback at 30°) and the G-suit not inflated. The mean blood pressure measured at the onset of the L-1 maneuver with subjects in an upright position and the anti-G suit inflated to 4 psi averaged 187 ± 3 mm Hg. These values were not significantly different. In addition, the peak mean blood pressure generated by subjects in a supine position (60° seatback) were not different from these levels. Thus generation of the L-1 maneuver by well motivated and trained subjects should permit tolerance of +5 to +6 Gz without the aid of the anti-G suit under acceleration conditions. However, within 5 sec, mean blood pressure fell by 30 - 35 mm Hg in both seatback configurations, regardless of the inflated anti-G suit. This amounts to a loss of 1 G tolerance. These data are interesting in view of the findings from previous studies. Wood et al (2) indicated that in an acceleration of +5 Gz, a venous pressure of 250 mm Hg at foot level would be required to maintain venous return in a relaxed (non-straining) subject. With relaxed subjects undergoing 30 sec of +4 and +5 Gz in a centrifuge, systolic blood pressure was involuntarily increased to nearly 200 mm Hg after the initial 5 sec of +G onset. Elevation of systolic blood pressure was due to increses in abdominal pressure, which approached 125 mm Hg at +4.5 Gz acceleration. This was possible because the abdominal contents behave like a hydrostatic system with the diaphragm acting as the upper surface (3). Alterations in the height of the diaphragm produce corresponding changes in abdominal pressure. It is also known that increases in airway pressure caused by forced expiration against a closed glottis produces simultaneous increases in abdominal and intrapleural pressure (4). These pressure changes are transmitted

to the peripheral systemic arterial system producing corresponding increases in systolic and diastolic blood pressures. Our results show a positive correlation between the changes in intrathoracic pressure and the changes in arterial blood pressure (see Figure 7). Maintenance of high intrathoracic pressures, as occurred during the L-1 maneuver in our subjects, and high intra-abdominal pressures began to impede venous return within 5 sec, and arterial blood pressure, while remaining elevated above resting levels, fell during the initial 5 sec of the maneuver. The physiological responses described in Figures 4 - 6 are characteristic of the changes that occur during sustained Valsalva maneuvers (see Figure 14). The decreasing venous return and fall in cardiac output are reflected by the decrease in pulse pressure and the compensatory increase in heart rate, as shown in Figures 3 and 5. The release of the L-1 is characterized by an increase in the pulse pressure and a decrease in heart rate. These responses occur as a result of baroreceptor induced reflexes, and support the findings and conclusions reported previously (2, 5). The fact that the mean blood pressure remained stable near 160 mm Hg and then recovered to about 180 mm Hg by the end of the 15 sec L-1 maneuver reinforces the importance of performing this protective straining maneuver in a consistent manner. This same conclusion was reached by Whinnery (6) who showed that only trained centrifuge subjects had a significantly higher G tolerance ($\sim$7 G compared to $\sim$5.5 G) when performing L-1 maneuvers.

Body position was shown to have a very pronounced effect on G-tolerance in relaxed subjects undergoing acceleration on a centrifuge (6). Without the aid of inflated anti-G suits, subjects in an upright position (at a 15° seatback angle) had a G tolerance of 3.13 $\pm$ 0.10 G but when subjects were in a supine position (at a 60° seatback angle), G tolerance was enhanced by 53% to 4.8 $\pm$ 0.16 G. When the anti-G suit was inflated, the same enhanced protection was

afforded by altering the body position. G tolerance went from 4.3 ± 0.06 G in the upright position to 6.2 ± 0.10 G in the supine position (7). This degree of enhanced protection is only evident when humans are subjected to increasing +Gz forces. It was found that only 3.7% to 3.9% of the total blood volume was displaced from the lower extremities into the central circulation when anti-shock trousers were inflated sequentially at either 40 or 100 mm Hg (8). This amounted to approximately 225 ml of blood and not the 750 - 1000 ml as previously suggested (9-11). This study (8) also showed that when the abdominal bladder of the anti-G suit was inflated, the volume of blood displaced into the central compartment increased to only 4.6% of the total blood volume, some 260 ml. Since there was no statistical difference in the blood volume displaced when the external compression on the lower extremities and abdomen was increased from 40 mm Hg to 100 mm Hg, we can project that the same amount of redistribution occurred in our subjects when the anti-G suit was inflated to 4 psi, some 210 mm Hg. At 1 G this small volume of blood redistributed into the general circulation may account for the lack of an effect on the pressor response seen during the L-1 maneuver in our subjects.

There was no indication that the effort of the L-1 maneuver became less intense as the experiment progressed, or that intercostal or abdominal muscular fatigue contributed to blood pressure and intrathoracic pressure changes in the different body positions. The electromyographic activity recorded from any of the groups of muscles we examined remained constant throughout the experiment whenever the L-1 maneuver was performed. There was no indication from the integrated (rms) signal that fatigue of any of the muscle groups was occurring as the number of times the L-1 maneuver was performed increased. Typically, as a contraction exerted at a submaximal tension becomes fatiguing, there is a progressive increase in the amplitude of the rms signal recorded from the surface EMG (12,13) and none of the EMG signals recorded from our subjects

showed this pattern. There was no indication that the EMG signal recorded during the last set of L-1 maneuvers was statistically higher or lower than those recorded from the initial set of maneuvers. It is also important to note that during the L-1 maneuver, no one muscle group was more dominant than another when the straining with whole body muscular tensing was performed. Altering the body position of the subject from a 70° spine-to-thigh angle to a 105° spine-to-thigh angle did not permit the use of larger versus smaller groups of muscles to predominate during the straining and tensing since the integrated EMG signals from the quadriceps and abdominals were the same under the four spine-to-thigh angles tested. This finding is also corroborated by the similar changes in the intrathoracic pressure measured during the different body positions. If one position had permitted greater use of the abdominal or intercostal muscles, then higher intrathoracic pressures would have been recorded, and this also would have been reflected by differences in the intra-arterial pressures recorded. By limiting the tension that muscles can generate during the straining maneuver to low levels, the compensatory effects of elevating the arterial blood pressure due to isometric muscle contractions (14-17) has been minimized. Many studies have shown that strength training will increase G tolerance (18-21), and that strength training was more effective than aerobic training. It has been suggested that the limiting factor for sustained G tolerance is the failure to maintain high levels of arterial blood pressure sufficient to provide adequate blood flow to the brain due to fatigue of the straining maneuver (2). Our data do not show that repeating L-1 straining maneuvers over a 2 - 3 hour experimental period leads to muscular fatigue. However, it must be kept in mind that all experiments were performed at 1 G and that the added exhaustive factor of performining the straining efforts under high G levels, as would occur with subjects on a centrifuge or engaged in aerial combat maneuvers, is not seen in

our experiments. The muscular tensing that is performed during the L-1 maneuver is a form of intermittent isometric muscular contraction. Tesch et al (21) pointed out that contractile failure of the leg muscles during aerial combat maneuvers could be one cause for pooling of blood in the exercising limbs and compromise venous return to the heart. Our data show that muscular tensing of the type that is performed during the L-1 maneuver without any chest fixation or elevation of intrathoracic pressure (and presumably abdominal pressures) can elevate the arterial blood pressure by about 35 mm Hg (see Figure 13). This would suggest that and increase of 1 G tolerance is achieved by this type of activity alone. Since our subjects were able to generate mean blood pressures that were almost 90 mm Hg higher than resting levels when performing the L-1 maneuver, the difference in the increment in blood pressure can be attributed to the generation of high abdominal and intrathoracic pressures. If these data are compared to the preliminary information presented in Figure 15 and Table 3, the effects of performing high tension isometric contractions simultaneously with the L-1 maneuver can be seen. Clearly, mean blood pressure was able to be maintained at a higher level, especially during the latter stages of the L-1 maneuver, when isometric handgrip contractions at 50% MVC were exerted together with the L-1 maneuver. The elevation and maintenance of blood pressure in response to this combination would project as being able to enhance G tolerance by another 1 G above that which is currently possible by the combination of anti-G suit inflation and L-1 straining maneuver alone. It is also important to note that for the one subject who performed handgrip contractions at an even higher tension (70% MVC), the elevation and maintenance of blood pressure was even higher, and would project to having a 2 G tolerance increment above that seen without the simultaneous handgrip contraction. The possibility that enhanced G tolerance could result during simulataneous isometric contraction of selected muscle groups with protective straining maneuvers and anti-G suit

inflation needs to be explored under conditions of increasing +Gz acceleration,

Conclusions

The data from this study indicate that generation of high blood pressures during L-1 straining maneuvers is not affected either positively or negatively by altering subject body position (i.e., from spine-to-thigh angles at 70°, 84°, 94° or 105°). This would suggest that alterations in the pilot seat configuration in current or future high performance aircraft for other reasons would not adversely affect the G tolerance of well trained aircrewmen.

REFERENCES

1. Shubrooks, S.J., and S.D. Leverett Effect of the Valsalva maneuver on tolerance to +Gz acceleration. J. Appl Physiol. 34:460-466, 1973

2. Wood, E.H., E.H. Lambert and C.F. Code Involuntary and voluntary mechanisms for preventing cerebral ischemia due to positive Gz acceleration. Physiologist 24: Supplement S-33 - S35, 1981

3. Rushmer, R.F. The nature of intraperitoneal and intrarectal pressures. Am. J. Physiol. 147: 242-249, 1946

4. Sharpey-Schafer, E.P. Effect of respiratory acts on the circulation. Handbook of Physiology Vol. 3 Amer. Physiol Society Bestheda p. 1875-1886, 1965

5. Laughlin, M.H., J.W. Burns and M.J. Parnell Regional distribution of cardiac output in unanesthetized baboons during +Gz stess with and without an anti G suit. Aviat. Space Environ Med 53: 133-141, 1982

6. Whinnery, J.E. G-tolerance enhancement: straining ability comparison of air crewmen, non aircrewmen and trained centrifuge subjects. Aviat. Space Environ Med 53: 232-234, 1982

7. Hrebien, L. and E. Hendler Factors affecting human tolerance to sustained acceleration. Aviat. Space Environ Med 56: 19-26, 1985

8. Bivins, H.G., R. Knopp, C. Tiernan, P.A.L. dos Santos and G. Kallsen Blood volume displacement with inflation of antishock trousers. Ann Emerg Med 11: 409-412, 1982

9. Kaplan, B.C., J.M. Civetta and E.L. Nagel The military anti-shock trouser in civilian pre-hospital emergency care. J. Trauma 18: 843-848, 1973

10. McSwain, N.E. Pneumatic trousers and the management of shock. J Trauma 17: 719-723, 1967

11. Civetta, J.M., S.R. Nussenfeld, and T.R. Rowe Pre-hospital use of military anti-shock trouser. JACEP 5: 581-587, 1976

12. Lind A.R. and J.S. Petrofsky Amplitude of the surface electromyogram during fatiguing isometric contractions. Muscle Nerve 2: 257-264, 1979

13. Petrofsky, J.S. and C.A. Phillips Discharge characteristics of motor units and the surface EMG during fatiguing isometric contractions at submaximal tensions. Aviat Space Environ Med 56: 581-586, 1985

14. Lind, A.R., G.W. McNicol and K.W. Donald Circulatory adjustments to sustained (static) muscular activity. In Physiological Activity in Health and Disease, ed K Evang, KL Anderson Baltimore, Williams & Wilkins pp 38-63, 1966

15. Lohrbauer, L.A., R.L. Wiley, S.J. Shubrooks and M. McCally Effect of sustained muscular contraction on tolerance to +Gz acceleration. J Appl Physiol 32: 203-209, 1972

16. Funderburk, C.F., S.G. Hipskind, R.C. Welton and A.R. Lind Development of and recovery from fatigue induced by static effort at various tensions J Appl Physiol 37: 392-396, 1974

17. Williams, C.A., J.G. Mudd and A.R. Lind The forearm blood flow during intermittent hand grip isometric exercise. Circ Res 48: Suppl 1: 110-117, 1981

18. Epperson, W.L., R.R. Burton and E.M. Bernauer The effect of physical conditioning on +Gz tolerance. Washington DC Aerospace Medical Association Preprints: 82-83, 1977

19. Spence, D.W., J.M. Parnell and R.R. Burton Abdominal muscle conditioning as a means of increasing tolerance to +Gz stress. Washington, DC Aerospace Medical Assocation Preprints 1981

20. Balldin, U.I. Physical training and +Gz tolerance Aviat Space Environ Med 55: 991-992, 1984

21. Tesch, P.A. , H. Hjort, and U.I. Balldin Effects of strength training on G tolerance. Aviat Space Environ Med 54: 691-695, 1983

TABLE 1 Anthropometric Data

Subject	Age (yrs)	Ht (cm)	Wt (kg)	Length of Leg[1]	Length of Thighs[2]	Length of Seat to Supra-sternal Crest	Length of Chest Supra-sternal crest to xyphoid notch	Length of Foot-plate
A	26	183	65	40.6	41.9	52.1	16.5	34.3
B	23	178	82	40.6	43.8	59.7	19.0	26.7
C	23	193	109	45.7	46.3	59.7	20.3	36.2
D	38	185	95	41.9	44.4	52.1	19.0	35.5
E	25	193	106	41.3	48.3	59.7	24.7	36.8
F	27	170	61	38.1	39.4	46.3	20.9	29.8
G	28	190	111	43.8	48.3	60.3	22.2	36.2
H	22	180	79	41.3	43.2	53.3	18.4	35.9
I	26	175	84	36.8	41.9	59.7	17.8	26.7
	26.4±1.6	183±3	88±6	41.1±0.9	44.2±1	55.9±1.7	19.9±0.8	33.1±1.4

1. Measured from lateral malleolus to the top of lateral condyle

2. Measured from trochanter to the lateral protrusion of the epicondyle of the femur

All lengths in cm

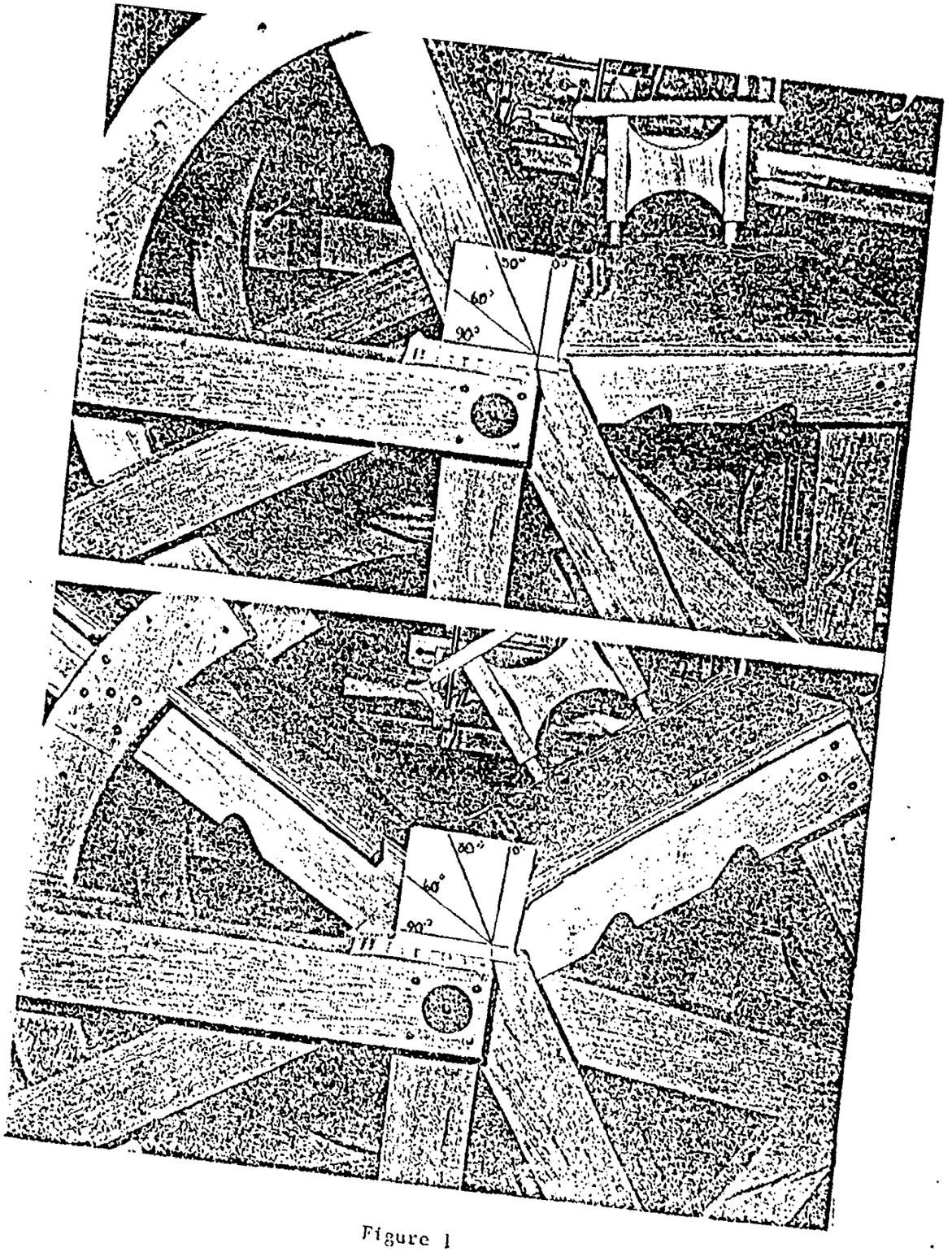
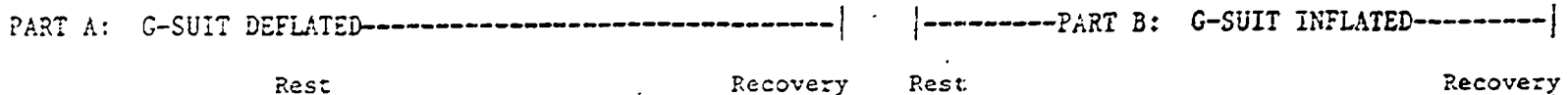


Figure 1

Pilot's Chair

FIGURE 2 EXPERIMENTAL PROTOCOL

EXPERIMENT 1



EXPERIMENT 2

15 sec L-1 straining maneuver

† Measure: HR
Esop
BP
EMG

* * G-suit inflated at start of maneuver, and deflated at end of maneuver

1-4 Refer to various spine-to-thigh angles: 70°, 84°, 94° and 105°

▲ Isometric hand-grip contraction at 40%MVC

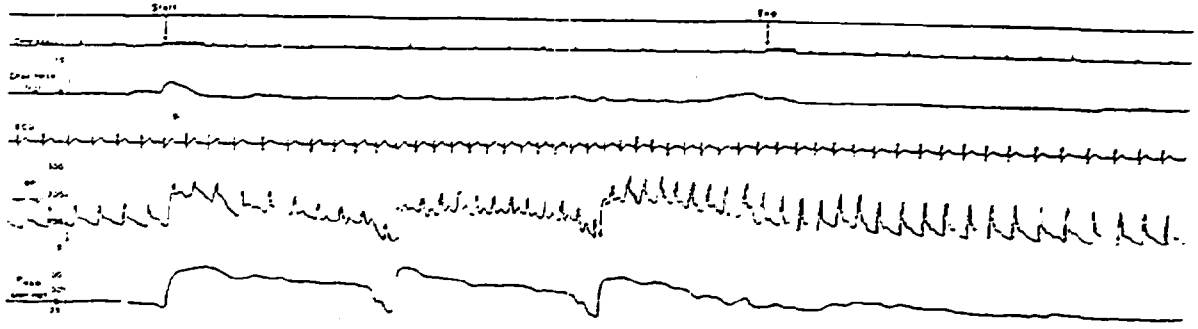


Figure 3

Tracing of Measured Physiological Variables

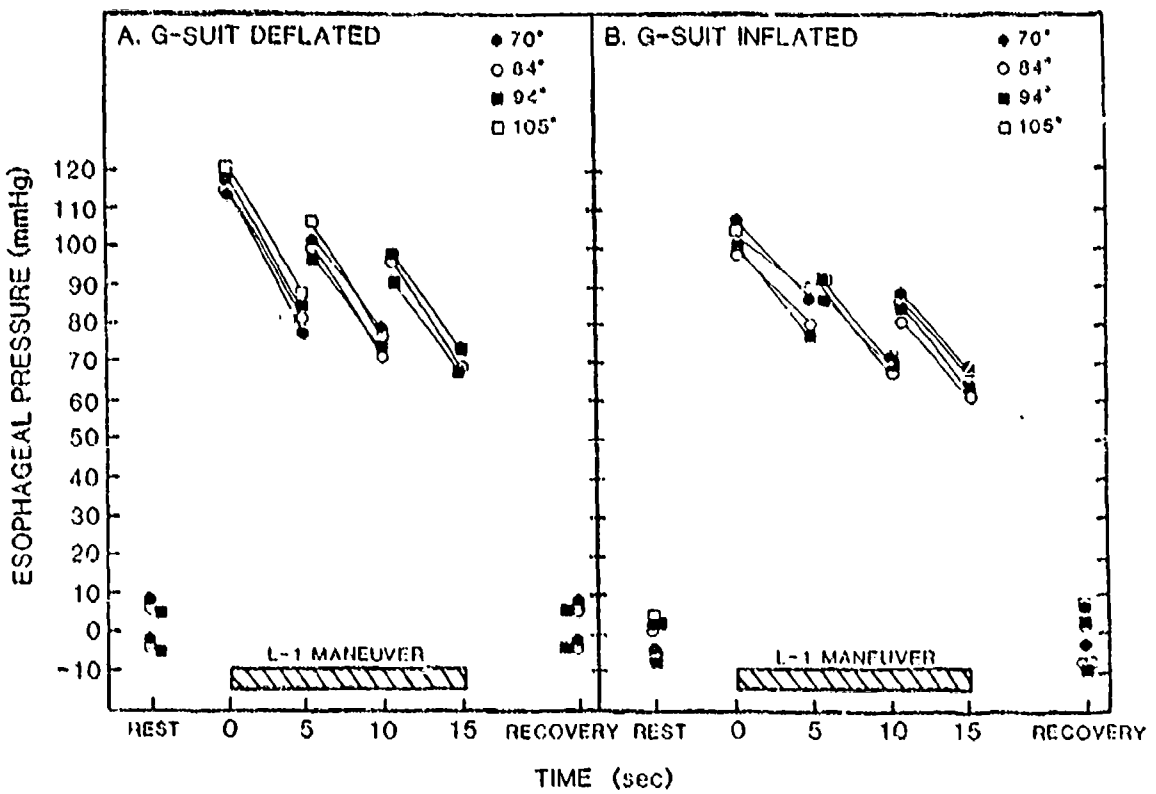


Figure 4

Change in Esophageal Pressure During the L-1 Maneuver

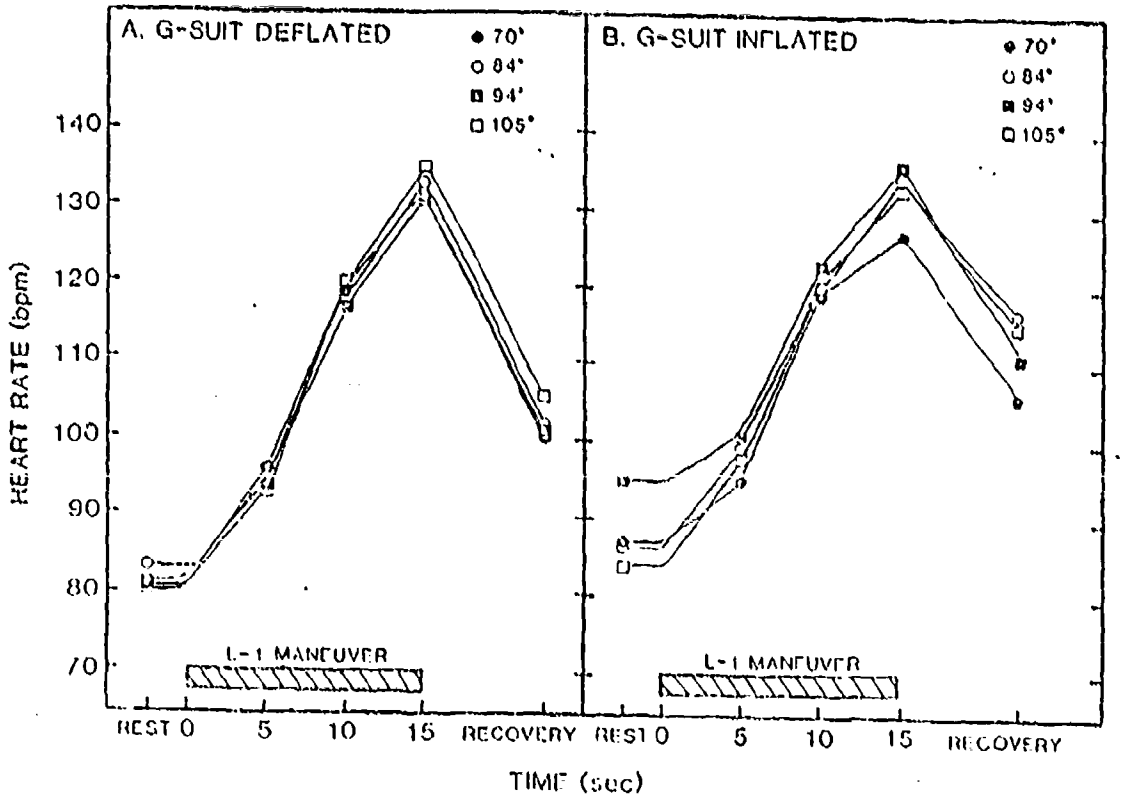


Figure 5

Change in Heart Rate During the L-1 Maneuver

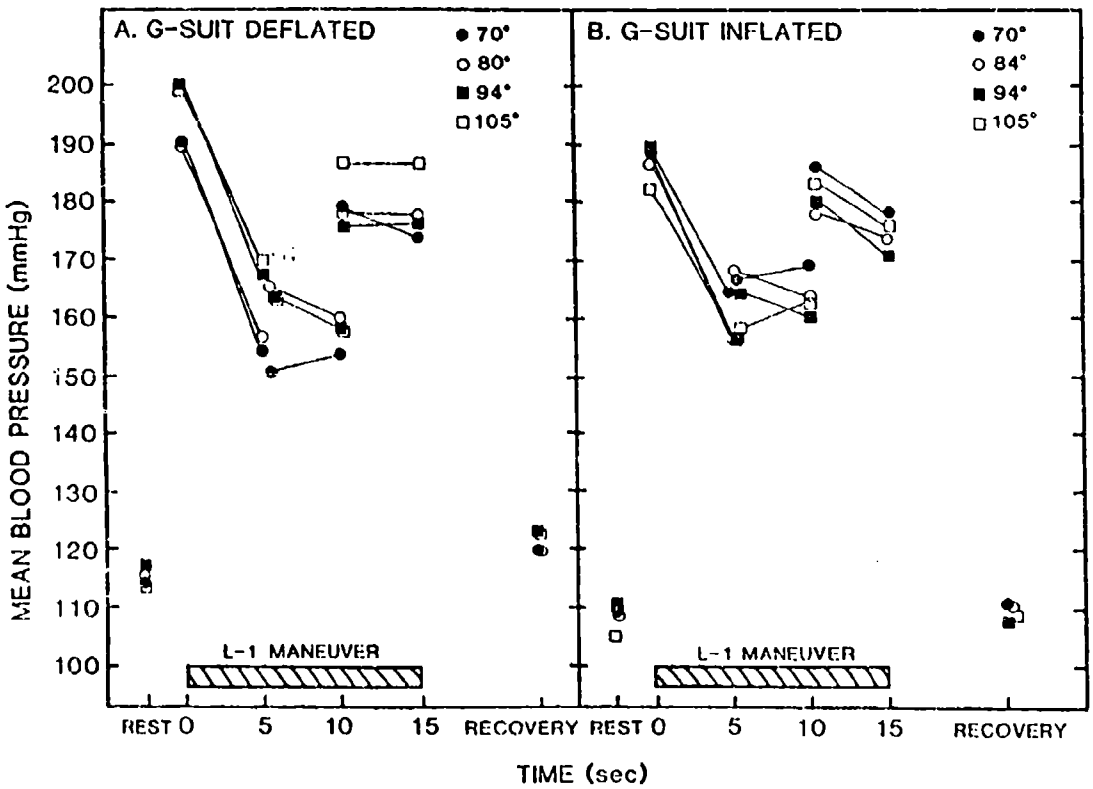


Figure 6

Change in Blood Pressure During the L-1 Maneuver

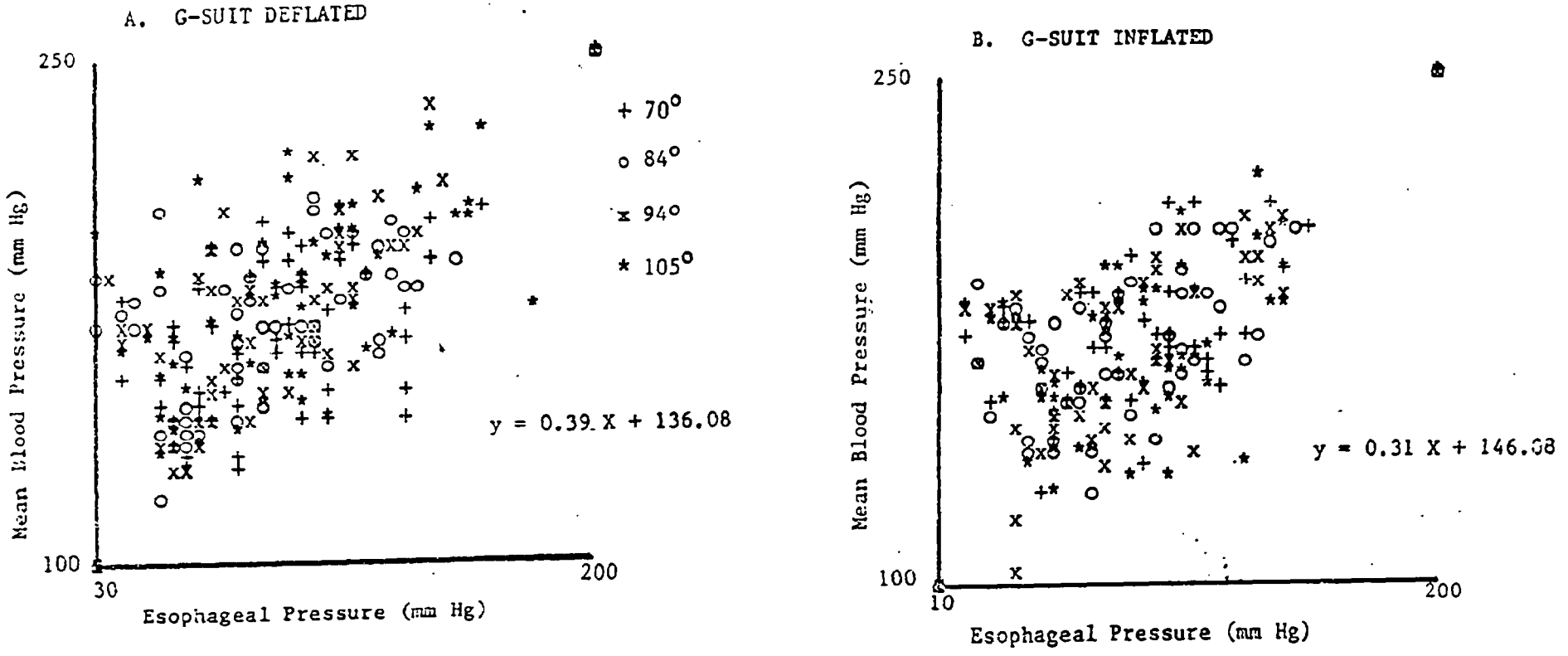


Figure 7

Relationship Between MBP and Esophageal Pressure

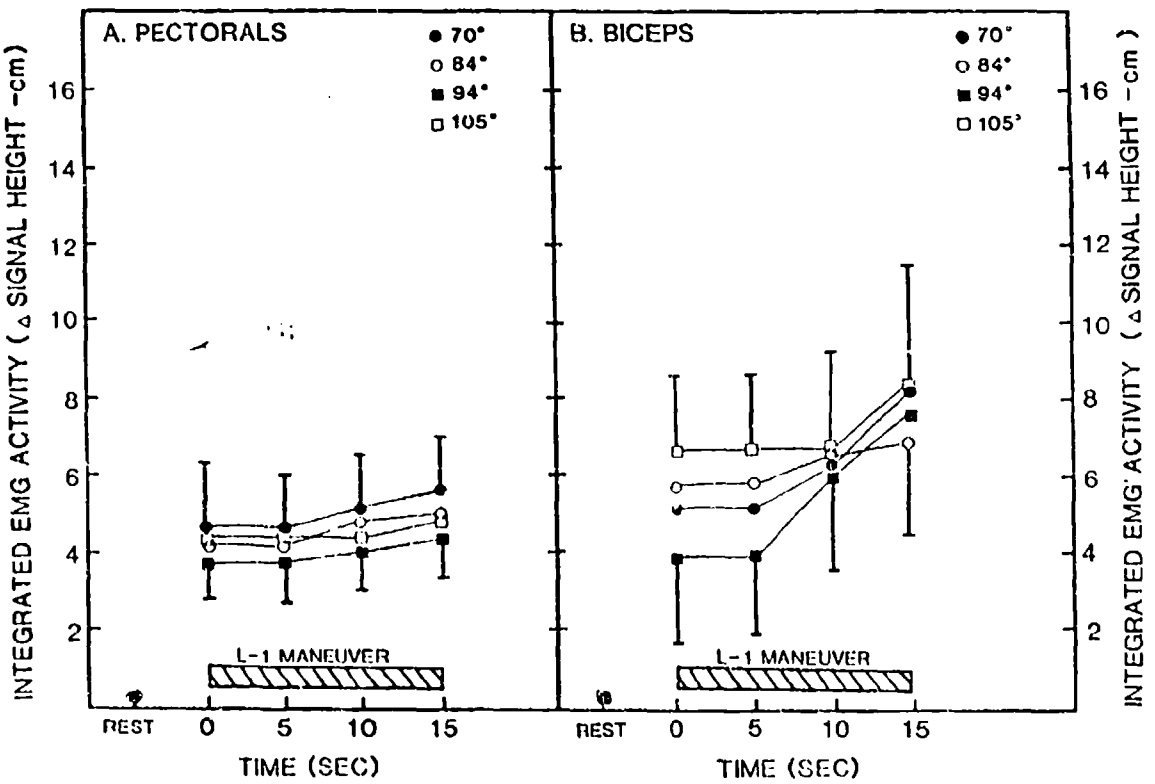


Figure 8

EMG Activity of Pectoral and Bicep Muscles

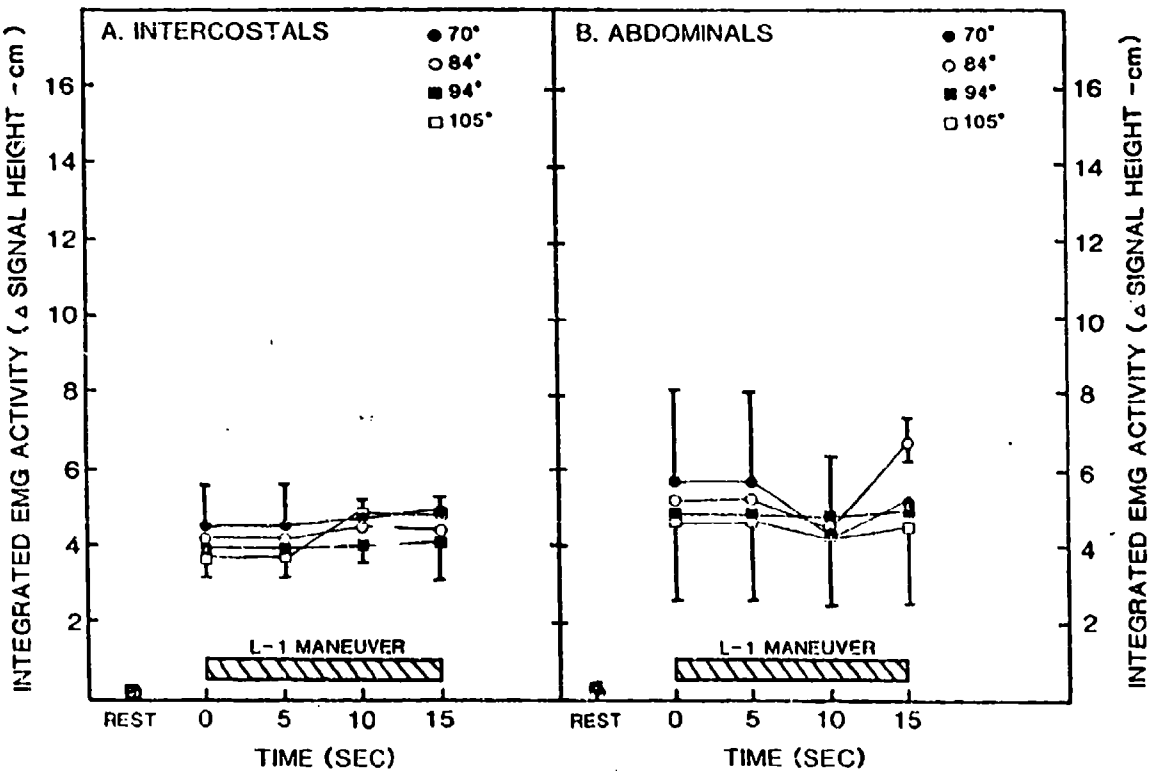


Figure 9

EMG Activity of Intercostal and Abdominal Muscles

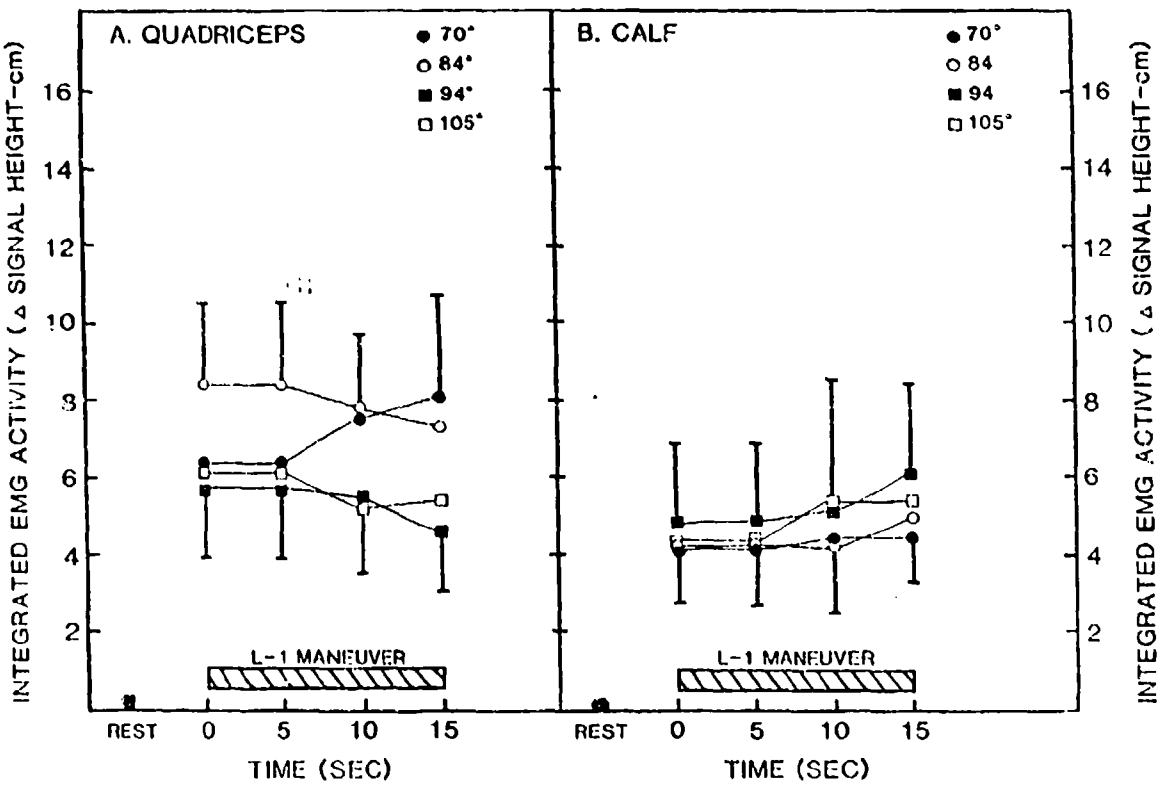


Figure 10

EMG Activity of Quadriceps and Calf Muscles

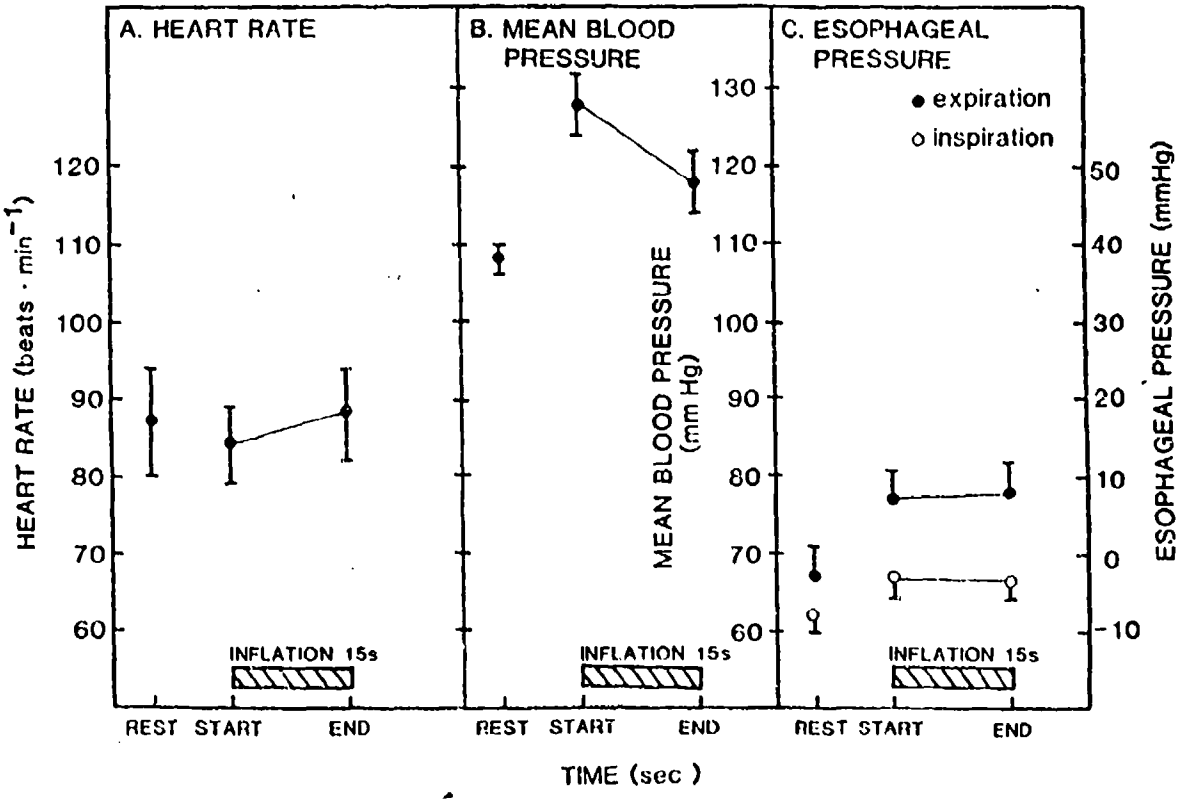


Figure 11

Effect of Anti-G Suit Inflation

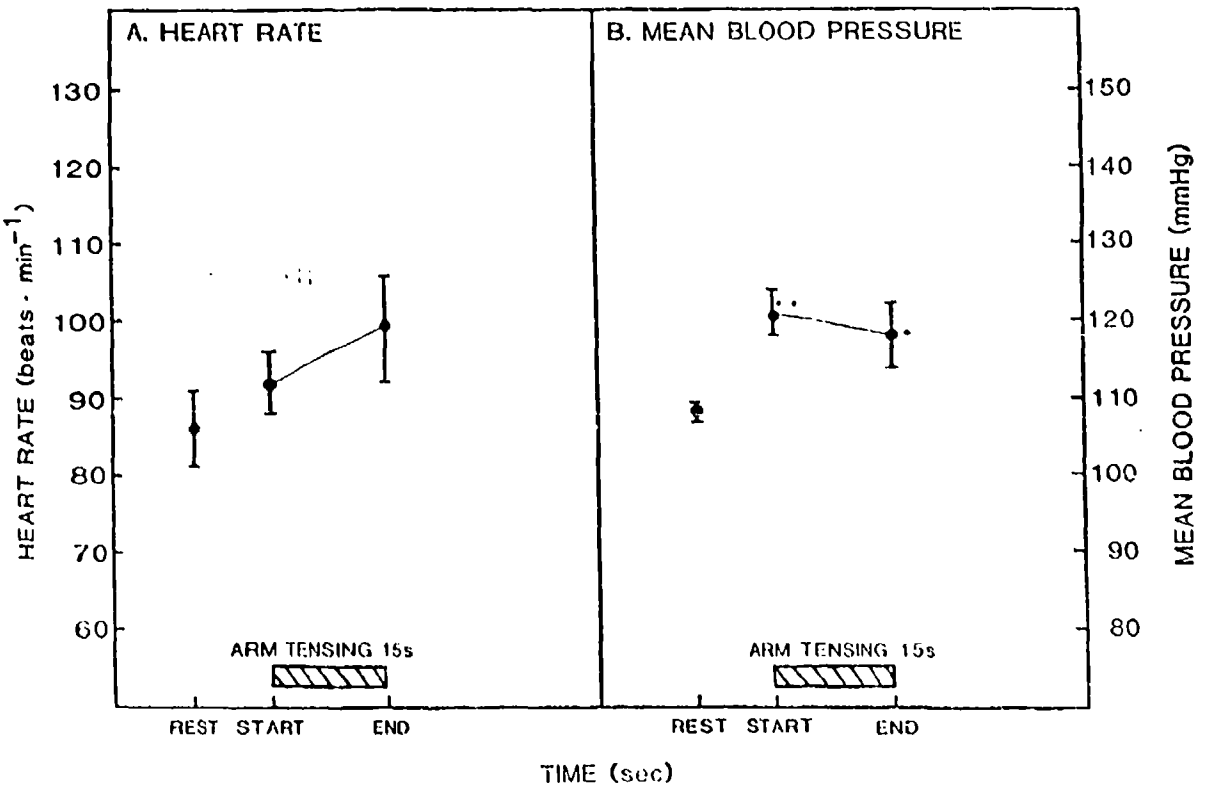


Figure 12

Effect of Arm Tensing Alone on HR and MBP

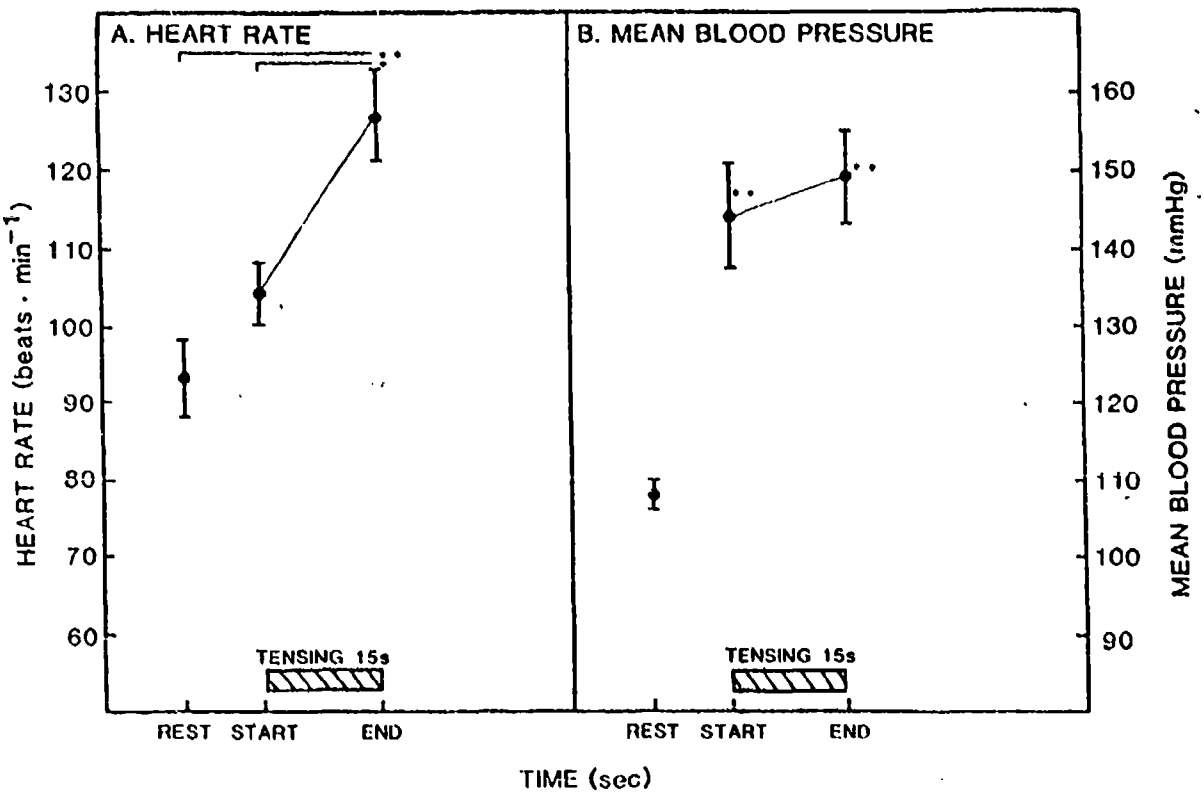


Figure 13

Whole Body Tensing Without an L-1 Maneuver

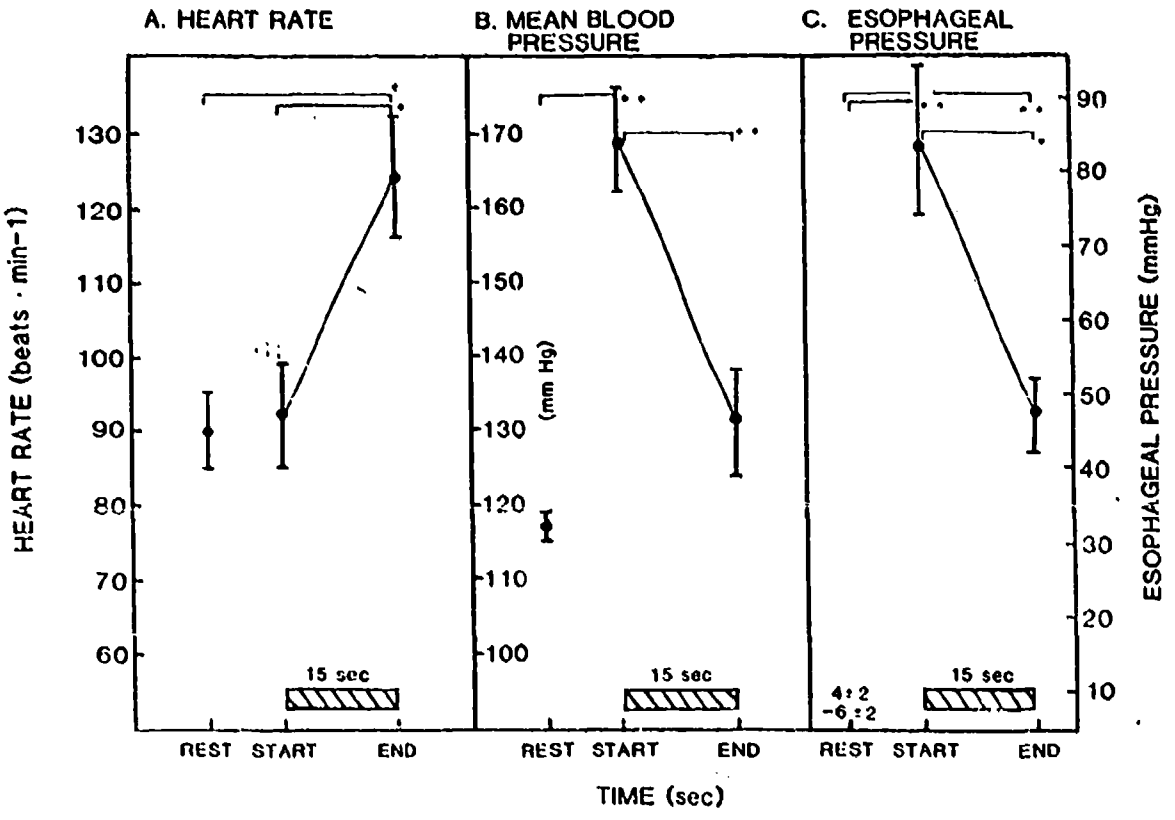


Figure 14

Changes During Valsalva

TABLE 2

Physiological Responses to L-1 Maneuver with Seatback at 60°

	Rest	0-5 sec	5-10 sec	10-15 sec	Recovery
HR (bpm)	89±5	101±5	122±5	137±7	103±7
MBP (mmHg)	104±2	171→145 ±4 ±5	148→150 ±4 ±6	1/0→172 ±10 ±8	114±4
Peso (mmHg)	5 →4 ±2 ±3	99→70 ±9 ±8	84→64 ±11 ±10	79→60 ±12 ±8	7 →2 ±3 ±3

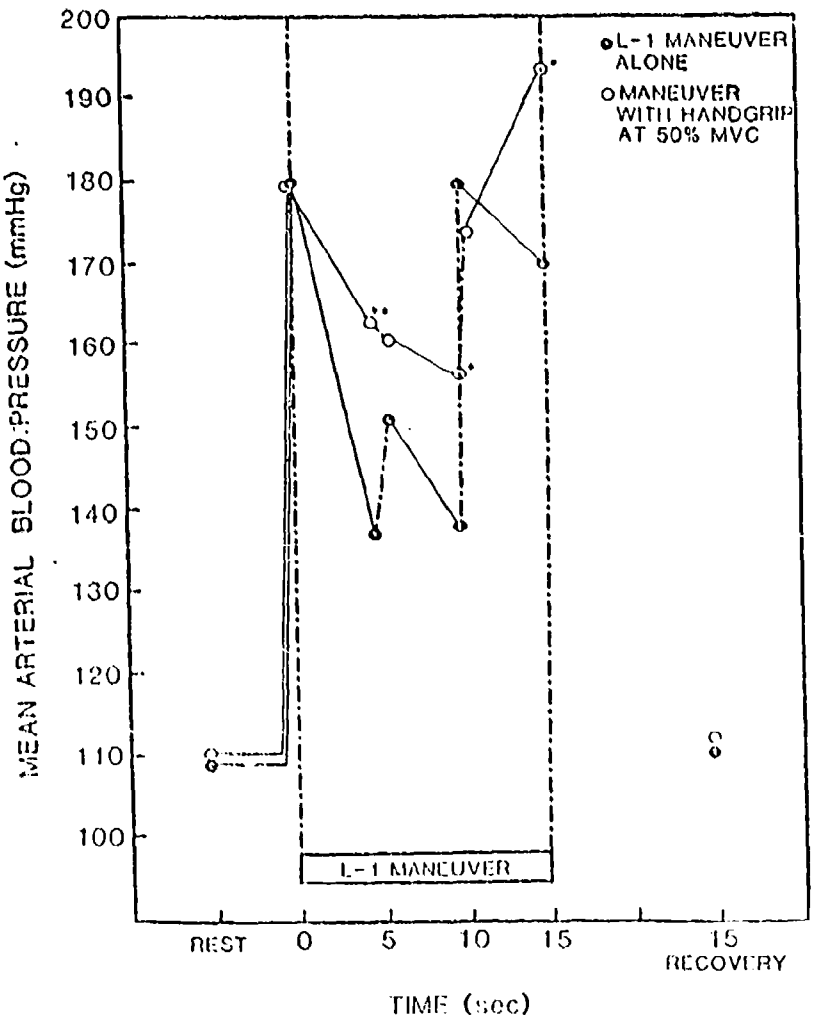


Figure 15

Changes in MBP During the L-1 and Fatiguing Handgrip

TABLE 3

Effect of Simultaneous Handgrip Contractions During L-1 Maneuvers on Mean Arterial Pressure

		During L-1 Maneuver			
	Rest	0-5s	5-10s	10-15s	Post 15s
Control	109 ± 2	180 → 137 ±4 ±7	151 → 138 ±9 ±10	180 → 170 ±9 ±5	111 ± 2
Handgrip at 30%MVC	111 ± 3	172 → 148 ±5 ±7	156 → 155 ±15 ±16	177 → 168 ±15 ±11	105 ± 2
Handgrip at 40%MVC	107 ± 3	164 → 159 ±8 ±7	167 → 152 ±2 ±1	170 → 170 ±5 ±5	106 ± 2
Handgrip at 50%MVC	110 ± 4	180 ‡→ 163 ** ±5 ±9	161 ‡→ 157 * ±8 ±6	174 ‡→ 194 * ±7 ±13	112 ± 3

‡ Not significantly different from corresponding control values (i.e. L-1 maneuver alone)
* P<0.01 from corresponding control values
** P<0.005 from corresponding control values

Note: data are averages ± S.D. for 4 subjects
data at different spine-to-thigh angles (70°, 83°, 94°, 105°) combined